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CASE REPORT

A case of intracoronary protruded thrombus caused after bailout stenting for side branch occlusion

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Summary A 69-year-old woman underwent percutaneous coronary intervention for a severe stenotic lesion in the bifurcation of the mid-left anterior descending artery and first diagonal branch. A single stent was implanted into the left anterior descending artery. After the stent strut was dilated by balloon inflation in the diagonal branch, dissection occurred at the ostium of the diagonal branch and resulted in side branch occlusion due to hematoma. Bailout stenting was performed in the diagonal branch, but thrombus projection occurred in the left anterior descending artery. Aspiration, balloon inflation and thrombolytic therapy were performed, but distal embolism developed. This case illustrates that thrombus projection caused by stenting in a side branch may occur as a rare complication in percutaneous coronary intervention.

Introduction

Intramural hematomas in coronary arteries are a well-known complication of percutaneous coronary intervention (PCI), and are identified by intravascular ultrasound (IVUS) in 6.7% of cases [1]. The main mechanism appears to be dissection into media without re-entry. In this case report, thrombus projection in the left anterior descending artery (LAD) occurred after stent implantation in the diagonal branch for the purpose of bailout. The thrombus was squeezed from an intramural hematoma in the dissected lesion of the diagonal branch. The case indicates that thrombus projection caused by stenting in a side branch may occur as a rare complication in PCI.

Case report

The patient was a 69-year-old female with a history of posterolateral myocardial infarction who

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Figure 1 CAG revealed severe stenosis of the bifurcation between the proximal portion of the LAD and the D1.

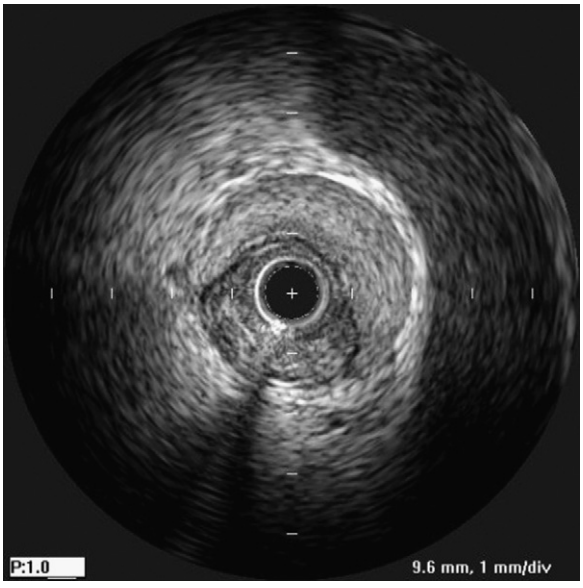

Figure 2 Pullback IVUS imaging from the mid-portion of the D1, showing a significant stenosis with an intramural hematoma in the D1.

was admitted for unstable angina. She had received drug treatment for hypertension and diabetes mellitus. Coronary angiography (CAG) revealed a total occlusion of the distal part of the left circumflex artery (Cx) and a severe stenosis in the bifurcation of the LAD and the first diagonal branch (D1) (Fig. 1). The right coronary artery provided good flow to the distal part of the occluded Cx via collateral arteries. The culprit lesion was thought to be a bifurcated lesion, based on the CAG findings. Therefore, PCI was planned for the bifurcated lesion. Aspirin (200 mg/day) had been administered for 3 years and ticlopidine (200 mg/day) was added 2 days before performance of PCI.

Heparin (5000 units) was injected immediately before catheterization and additional heparin (1000 units) was then administered every hour. A guidewire was crossed through the stenotic lesion to the LAD and another wire was extended to the D1. IVUS examination of the LAD showed that the bifurcation of the LAD was narrow due to an atheromatous plaque, and that atheromatous plaque of moderate volume existed in the LAD site where the D1 had just arose. However, the D1 ostium was not severely stenotic, in contrast to the CAG findings. Therefore, we chose a strategy of single stenting in the LAD. A stent (Cypher 3.5/23-mm, Cords, Johnson & Johnson, Miami Lakes, FL, USA) was implanted in the LAD after covering the D1 ostium, while the wire remained in the D1 for protection. After that, the D1 ostium severely narrowed due to a plaque shift from the LAD. A new wire was advanced into the D1 through the struts of the sailed D1 and the protective wire in the D1 was pulled back. Then, balloon dilatation with a 2.5-mm balloon catheter was performed at the D1 ostium jailed by the struts. Immediately afterwards, a dissection occurred from the D1 ostium just near the struts and extended distal to the D1 to occlude the D1. A meticulous IVUS examination of the D1 showed intramural hematoma extending from the D1 ostium to 2 cm distal to the D1 (Fig. 2).

To resolve the acute occlusion without stenting to the dissection, long inflations of a balloon for tacking the dissected wall up and over-dilatations of balloons to the distal region, and stinging of the guidewire from the false lumen to the true lumen for making a re-entry into the dissection were attempted. However, bailout was not achieved after performing these procedures for 2 h. Finally, implantation of a stent (Cypher 3.0/33-mm) in the D1 as forming reverse T stenting was performed, but this created a mass that projected into the bifurcation (Fig. 3). An IVUS examination of the LAD showed that the mass was a thrombus (Fig. 4). We aspirated the thrombus using an aspiration catheter, compressed it by balloon dilatation, and poured urokinase (960,000 units) and argatroban (10 mg) directly into the thrombus using a pulse-spray infusion catheter for thrombolysis (UltraFuse, Boston Scientific, Natick, MA, USA). The procedure reduced the size of the projected thrombus in the stent and resulted in a small embolism in the distal LAD. Although small myocardial infarction occurred because of distal embolism, the clinical course of the patient after the procedure has been good.

Figure 3 Implantation of a stent in the D1 caused projection of a mass into the bifurcation of the LAD and D1.

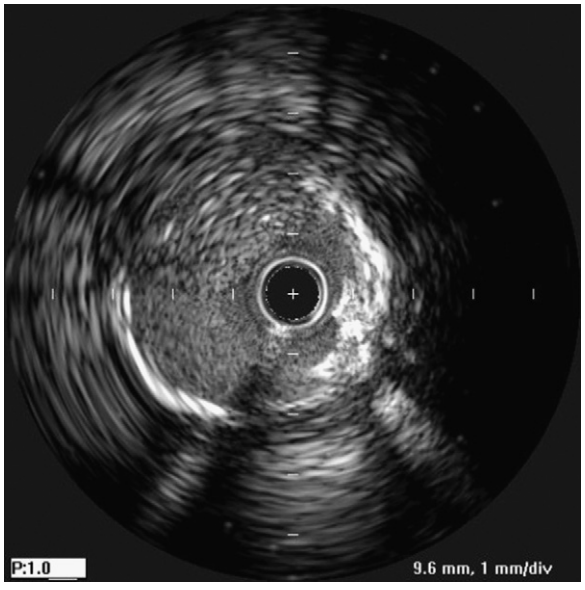

Figure 4 IVUS imaging of the LAD. Projected thrombus was observed in the D1 ostium and just proximal of the D1 bifurcation.

Discussion

Stenting of a bifurcation lesion carries a risk of side-branch compromise [2—4]. The risk is increased if there is an eccentric lesion at the bifurcation site, a stenosis in the ostium of the side branch (true bifurcation lesion), or a narrow angle between the vessels [4,5]. The mechanisms of side-branch compromise are usually plaque shift and/or a dissection extending into the side branch [5].

In the current case, the luminal narrowing of the D1 ostium that occurred immediately after stent implantation in the LAD was due to plaque shift, and the cause of the D1 narrowing after balloon dilation at the ostium was a dissection. The cause of the new narrowing in the bifurcation after stent implantation in the D1 was certainly a thrombus, which was squeezed out of the hematoma by stenting in the false lumen of the dissection. It has been reported that an extension or growth of an intramural hematoma occurs proximally and tends to narrow the ostium of another vessel without stenotic lesions; however, the cause of the narrowing is usually a hematoma moving in the media [6,7]. In contrast, the projection of a thrombus, as observed in our case, is very rare.

Coagulation of blood in the intramural space without re-entry depends on the time from the occurrence of the dissection or the amount of tissue factor in contact with blood in the intramural space. In the current case, successful bailout stent implantation in the D1 required 2 h, and the long duration may have led to thrombus formation; however, the time required for blood to coagulate in media is uncertain.

Avoidance of movement of a thrombus from the dissected space to the bifurcation when placing a stent in the D1 is difficult. There is free space in T stenting of a bifurcation, and in our case it was impossible to cover the entry close to the ostium of the D1. Even if reverse crush stenting of the D1 is performed, the struts of the stent in the LAD may prevent the struts of the stent in the D1 from attaching closely to the wall of the D1 ostium. Furthermore, even if the stent is closely attached to the artery wall, it may not close the entry completely and the thrombus may be squeezed out of the entry through the strut space. Our case indicates that thrombus projection caused by stenting of a side branch is a rare complication of PCI that can occur due to side branch occlusion caused by a hematoma accompanied by a long bailout period.

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